REMOTE EFFECTS OF BRAIN TRAUMA.

(Symptoms, Course and Prognosis.)

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Naturally in this article acute abscess, acute pachymeningitis or leptomeningitis, mental effects of sudden compressions or the acute traumatic apoplexies are excluded. We have here to deal with late abscess or cyst formation, localized sclerosis, post traumatic epilepsy, psychasthenia, hysteria and the traumatic psychoses. These conditions are of great importance in connection with workingmen's compensation, chiefly because the onset of the permanent symptoms is usually weeks or months after the injury, and in the interval while resting from his usual work the patient may be apparently well. These cerebral defects resulting after injury are even more difficult of a fair interpretation than are the spinal cord disorders which also occur late and after a free interval. In cerebral injury and its remote effects we have to consider the personality of the patient, before the injury, e. g., the inherent defects and peculiarities which are aggravated by the trauma and the probable duration of the neurological signs which have become more evident since the injury. Tabes and general paresis or cerebrospinal lues may have existed in latent degree previous to injury and become aggravated by the shock or the other added effects of the trauma. The same may be true of epilepsy. A traumatic psycho-neurosis is a real thing and has a direct relationship to trauma and yet because of previously existing psychic defects it is difficult to estimate just how much the resulting disability can be attributed to the accident. For example, a neurasthenic or hysterical constitution may have added to it by trauma a distinct psychasthenia syndrome which is of indefinite duration and doubtful prognosis because of the pre-existing make-up of the patient. This difficulty is equally true of the traumatic psychosis in which there are many other possible contributing factors of causation to be considered.

ABSCESS AND HEMORRHAGIC CYST.

The onset of symptoms of abscess may be from three to five weeks after the head injury, but not infrequently, the interval is months and even years. English reports a case with autopsy ten years after the injury. During the interval the patient may suffer from indefinite subjective symptoms referable to the head, such as headache of variable character, attacks of numbness or tingling of one limb, temporary attacks of aphasia or recurrent convulsions, before any of the symptoms of infection appear. Later, acute symptoms, such as fever, slow pulse, vomiting, optic neuritis and paralysis, referable to focal lesions, occur. Of these symptoms, optic neuritis is the most important, being often unilateral and corresponding to the side on which the abscess is situated. There is a characteristic mental state of apathy and somnolence with periods of restlessness and confusion.

The course of these symptoms is progressive but often very slow, death resulting from chronic toxæmia or from bursting of the abscess into the ventricles.

The diagnosis has to be made from epilepsy, a psychosis, or tubercular meningitis.

What has been said of abscess is largely true, also of post-traumatic cysts which are the result of hemorrhage and softening of brain tissue. The course is, however, less progressive, the symptoms more focal and less general and the result a chronic epilepsy of Jacksonian type frequently, although general convulsions may also be the result of a focal lesion. Susceptibility to alcohol is increased by all head injuries of serious degree and the irritative effects of a cyst formation may be enhanced or produced by a moderate amount of alcohol.

TRAUMATIC EPILEPSY.

Aside from the Jacksonian form of epilepsy due to focal lesions of a hemorrhagic-cystic nature or to scar formation, epilepsy of the same type as the idiopathic form can result from brain trauma even when no symptoms or signs relative to a focal lesion can be demonstrated. In such cases one has to distinguish between a pre-existing epileptic status and true traumatic epilepsy and to do this a very careful history dealing with symptoms of masked epilepsy such as psychical aberration or petit mal attacks or undue susceptibility to alcohol must be elicited. Where such symptoms are present in the previous history, the relation of trauma to the epilepsy must be given a position of secondary importance. There is no way of deciding the degree of importance of this relationship, except from the personal history, as the signs and symptoms of traumatic epilepsy and "idiopathic" epilepsy are the same in numerous cases. It is only when we have signs of focal brain irritation or destruction that we can be sure of the cause being traumatic. Furthermore, it should be remembered that epilepsy of traumatic origin may manifest itself either in Jacksonian or generalized attacks or very late after the trauma. For example, English found 21 cases out of 300 which developed after a year, and of these only seven were of the Jacksonian type. The statistics of the Craig Colony for Epileptics show that out of over 800 cases, in only one was there evidence of a fracture of the base of the skull, showing how many cases there are in which the gross injury is very slight. In many cases we must regard the head injury as only a spark which ignites a pre-existing epileptic constitution and this is especially true of an alcoholic patient.

It should also be noted that many cases may be the result of fright and the attacks be really hysterical in nature. To distinguish between true epilepsy and hystero-epilepsy is not always easy, especially if the actual attack is not observed. None the less, hysterical epilepsy is a real disease and just as incapacitating as true epilepsy but the prognosis is infinitely better.

While the time elapsing between the injury and the onset of epilepsy is not a definite guide to diagnosis, we may consider an epilepsy arising

many years after the trauma as not altogether due to it, for in persons past middle life arteriosclerosis or cerebral syphilis may be the real basis for the attacks. Traumatic factors figure as causes chiefly in youth.

When the attacks are Jacksonian in nature from the beginning, and other causative factors, such as cerebral syphilis or alcoholism can be definitely ruled out the relation of trauma to the attacks is fairly certain even if the attacks begin months after the injury and even if there has been no fracture of the skull at the time. Most attacks have begun within one year from the date of injury but there have been notable exceptions to this rule, e. g., a case reported by Lloyd and Deaver, in which five years after an injury a young man of twenty-one developed Jacksonian attacks; his skull was opened and neither fracture nor gross evidence of brain injury could be found.

TRAUMATIC PSYCHASTHENIA.

This is a condition of real disability due chiefly to headache, dizziness, irritability and abnormal susceptibility to fatigue, not differing in character from symptoms of ordinary neurasthenia of the primary form. Such patients are often unjustly accused of malingering. The headache in these cases is the most distressing of the symptoms and the most unresponsive to therapy. It may be due to bony exostosis, meningeal adhesions or to slight luxations of cervical vertebræ. More often the headache like the other symptoms is the result of some minute change in the cortical cells (possibly the result of the œdema which follows concussion) impossible of demonstration except by the history and symptoms and the improvement which gradually occurs when the patient is made free from responsibility and anxiety over the outcome of his condition. Such cases under rest and diversion and abstinence from stimulants, will recover in one or two years, provided there is no antecedent neurasthenic or psychotic taint.

HYSTERIA.

This disorder, when of traumatic origin solely, is perfectly recoverable, but its duration is apt to be very uncertain and somewhat dependent upon the confidence the patient has in his physician, and the earlier the diagnosis is made, i. e., before all sorts of other diagnoses have been suggested. As was said above, it may be very difficult at times to distinguish between real epilepsy and the hysterical form. The other manifestations of hysteria may lead to erroneous treatment which prolongs unnecessarily the period of disability, i. e., unnecessary operative procedures for hysterical pain, etc.; or the conditions may be aggravated by too much therapy of another sort, e. g., plaster casts for hysterical joints.

The instillation of confidence in the future outcome, and the education of the patient combined with rational suggestive psycho-therapy, will do much to clear up the diagnosis as well as to shorten the period of disability which is real enough, even though hysterical. In this connection we must refer to the form of "traumatic" hysteria which subsides promptly upon the settlement of compensation. In Germany, by reason of intensive industrial legislation, this so-called "pension hysteria" has developed to a greater extent, perhaps, than in many other countries. This seems to be by reason of the German method of paying compensation, which continues during the period of disability, while other countries have a cash settlement basis. Thus, in Denmark the percentage recovering from traumatic neuroses is 93.6. In Germany it is only 9.3 per cent.

TRAUMATIC PSYCHOSIS.

The difficulty in assigning to trauma the leading or only role in the causation of any form of insanity is obvious. There are so many other factors concerned, e. g., alcoholism, syphilis, arteriosclerosis, epilepsy, and the inheritance of insanity. We are also confronted again with the fact that a true traumatic psychosis may occur very late after the injury. Pierce Bailey concludes from an analysis of the State Hospital statistics of New York, that trauma is an actual cause in less than one per cent. He bases this conclusion from the small percentage of instances of fracture of the skull in the histories of these asylum patients. However, there is, none the less, a true form of traumatic insanity of favorable prognosis in most instances, but of very uncertain duration. Its symptomatology is fairly definite and sets off the disorder from other more chronic forms of insanity as well as from other temporary forms.

Adolph Meyer has made the most complete study of this disorder on record, both from the literature and from personal observation of cases. He recognizes a true primary traumatic psychosis following definite head injury, and characterized in most cases by "a protracted delirium, partial disorientation, i. e., variations between clearness and haziness of the sensorium, a certain prominence of fabrications of dream-like situations, further difficulty of ready remembrance and calculation."

His cases are minutely described and analyzed. The main facts to be emphasized from an analysis of these cases and from other similarly studied are the following:

1. That the lesions are usually gross and productive of secondary degenerative changes, the latter being enhanced probably by the nutritional disturbance due to œdema.
2. That fracture of the skull or other external signs are infrequent.
3. That subsequent tumor formation, especially bony spurs of the endocranium, resulting from organized clot, may be the cause of continued symptoms after those typical of primary traumatic psychosis have disappeared.
4. That recovery from the psychosis, other things being equal, can be confidently predicted and the duration is usually about two months.
5. That "after effects" appear in many cases, characterized by marked irritability, forgetfulness and very distressing sensations in the head of a burning, creeping character or ordinary severe headache and dizziness aggravated by alcohol, to-

bacco, emotion or posture (vaso-motor instability). Also by slowness of thought, easy fatigue and inability to keep impressions. These symptoms disappear in time, but may last a year or more. If they do not disappear there are other complications, e. g., arteriosclerosis, not directly due to the trauma.

It is because of these comparatively mild after effects that such patients are often thought to be malingering. But the symptoms are so uniform and are demonstrated so consistently that a little observation of the patient under ordinary circumstances should soon decide this point. Such patients are reluctant to take part in any social activity; feel better when left alone; are incapable of sustained work or play and show a very pronounced irritability and also a marked degree of "absentmindedness," i. e., they easily forget instructions, messages or names, a disorder of attention being largely responsible for these "memory" defects. However, it is in these cases that one needs to be on guard against failing to distinguish a beginning general paresis so that in all such instances a lumbar puncture is warranted.

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A REVIEW OF SOME OF THE LATER DEVELOPMENTS ALONG IMMUNOLOGICAL LINES *

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In reviewing recent literature we are impressed with the fact that we are entering a new era in the study and treatment of infectious diseases.

When the vaccine therapy was first introduced by Wright and his co-workers we were quite enthusiastic, forming at that time strong opinions on specificity and believing that all infectious diseases should be treated solely upon a specific basis. Later studies, however, and taking into consideration the work of Abderhalden, Erhlich, Vaughan, Jobling, Petersen and others our opinions as to specificity have undergone decided modifications, so that even though we still believe specificity must be observed in following out vaccine therapy in the treatment of infectious diseases, we do not believe that specific treatment is the sole or even the main factor.

A review of some of the later developments along immunological lines shows that of all the numerous elements which enter into the physio-biological system of balanced reactions the most interesting from a physiological and pathological standpoint is the relationship existing between the proteolytic serum ferments on the one hand and the serum anti-ferments on the other. Unquestionably the increase or decrease of these elements has a vital influence upon normal metabolism.

It appears to be a well established fact that under normal conditions there is, and must of necessity be, a balanced relationship between these elements, and that any condition that interferes with this relationship will upset the normal metabolistic order of things and pathological changes due to functional deviation will inevitably be the result. If this be true, and there seems to be ample justification for this belief, then the big problem confronting the medical world today is how to control or maintain this normal balance between the ferments and the anti-ferments by increasing or decreasing either one as occasion requires.

It was formerly thought, that following the parenteral introduction of a bacterin or so-called vaccine, specific bactericidal or bacteriolytic substances were produced, whose function it was to attack the homologous invading bacteria, destroying them by the process known as lysis. As a result of this lytic action toxic end products became available and these toxic end products being specific, that is having a combining affinity for highly specialized cellular elements designated by Erhlich as cell or fixed receptors, become anchored to the cells, through the media of these receptors causing injury to the cells, which injury may be partial or complete, depending upon the amount of toxin available and the amount so anchored. If cells so attacked are not injured to the point of destruction they may undergo regeneration and during the process of such regeneration the cells provide against injury in the future from similar attacks. This protection is accorded by the formation of cell receptors or toxic combining elements in excess of the capacity of the cells. As a consequence this surplus is secreted or excreted into the surrounding body fluids becoming free receptors or anti-toxins whose mission it is to combine with the specific toxic elements before they have the opportunity of coming in contact with the cells. It was supposed in this way specific immunity became established.

However, it is now a well-recognized fact admitted even by Wright and his co-workers, that the therapeutic or immunological results, following the parenteral injection of bacteria, are not explained wholly on a specific basis as certain facts indicate that non-specific benefits result from bacteriotherapy. The possibility that the explanation for this effect is to be sought in the mobilization of non-specific ferments must be taken into consideration.

While the introduction of bacteria parenterally does, beyond question, stimulate the production of specific bacteriolytic substances and incidentally specific anti-toxic bodies, they do not apparently stimulate the production of specific proteolytic substances. It would seem logical in the light of recent developments and knowledge that as a result of bacteriolytic action upon the bacteria, substances of an-anti-tryptic character become available. These substances possess the property of inhibiting anti-ferments, thus removing all opposition to the normal ferments, permitting autolysis to take place and the consequent liberation of toxic end products which represent the result of cleavage of the individual's own protein elements.

In this connection we must bear in mind that

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